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ORIGINAL ARTICLE

Echocardiographic effect of successful balloon mitral valvuloplasty on right ventricular function

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KEYWORDS

Mitral stenosis;
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Abstract *Background:* Rheumatic cardiac disease is an immunologic phenomenon that may affect any of the heart valves and is by far the most common cause of mitral stenosis.
Aim of the work: To evaluate impact of successful percutaneous balloon mitral valvuloplasty on right ventricular function.
Subjects and methods: 30 consecutive patients who underwent balloon mitral valvuloplasty for rheumatic mitral stenosis between September 2010 and July 2011 at Ain Shams University hospital were included.
All patients were subjected to transthoracic echo Doppler study, tissue Doppler imaging and TEE for: (A) Assessment of severity of mitral stenosis. (B) Peak myocardial velocities during systole, early and late diastole. (C) Assessment of RV function by measurement of TAPSE and Tie index.
Results: The study revealed a significant drop in TAPSE (before and 24 h after, *P* 0.008; before and 3 ms after, *P* 0.001) respectively. There was no significant change 24 h and 3 ms after the procedure (*P* 0.220) .There was no significant change in IVC flow before and after 24 h (*P* 0.221). There was no significant drop between before and after 3 ms (*P* 0.062). There was no significant change between 24 h and 3 ms after the procedure (*P* 0.264). There was a significant drop in Tie index (before and 24 h after, *P* 0.008; before and 3 ms after, *P* 0.009) respectively but no significant drop was found 24 h and 3 ms after the procedure (*P* 0.373).
Conclusion: The current study showed a significant improvement in both systolic and diastolic functions of RV as observed by different echocardiographic parameters post BMV in patients without organic TV disease.

1. Introduction

Rheumatic cardiac disease is an immunologic phenomenon that may affect any of the heart valves and the myocardium and is by far the most common cause of mitral stenosis.[1]

Mitral stenosis leads to increase in left atrial pressure which results in a passive rise in pulmonary venous and arterial pressures.[2] It eventually leads to right ventricular dilatation and

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failure. Thus, right ventricular dysfunction is an important indicator to evaluate the severity of mitral stenosis.[3]

The quantitative echocardiographic assessment of right ventricular function is difficult because of the ventricle's complex trapezoidal anatomy and narrow acoustic window.

Two dimensional echocardiography is the mainstay for analysis of RV function, but recently alternative techniques have been proposed, including tissue Doppler imaging (TDI) techniques, three dimensional echocardiography, magnetic resonance imaging (MRI), and even invasive assessment of pressure–volume loops.[4]

The morphologic information provided by echocardiography is important for evaluating potential candidates for percutaneous balloon valvuloplasty.[5]

2. Subjects and methods

Thirty consecutive patients who underwent balloon mitral valvuloplasty for rheumatic mitral stenosis between September 2010 and July 2011 at the Ain Shams University Hospital were included in this study. All patients who were included in the study had formal written consent.

2.1. Inclusion criteria

- Sinus rhythm.
- All the patients were in New York Heart class $\geqslant$II or $\leqslant$IV.
- Moderate to severe MS (i.e., a mitral valve area $\leqslant 1\ cm^2/m^2$ body surface area (BSA) or $< 1.5\ cm^2$ in normal-sized adults.[6]
- Suitable valve morphology by echocardiographic criteria.
- Absence of concomitant cardiovascular disease requiring surgical correction.

2.2. Exclusion criteria

- Atrial fibrillation.
- Systemic hypertension.
- Diabetes mellitus.
- More than mild mitral or aortic regurgitation and/or aortic stenosis.
- Lung diseases.
- Pulmonary valve disease.
- New York Heart Association functional class IV.
- Previous aortic or mitral valve surgery.
- Echocardiographic criteria for contraindications of balloon valvuloplasty e.g. mitral regurgitation grade III or IV, left atrial thrombus, heavily calcified mitral valve annulus, commissural calcification and heavy subvalvular affection).[7]
- Organic tricuspid valve affection.

2.3. Echocardiographic measurement

- All patients were examined in the left lateral decubitus position by M-mode, two-dimensional, Doppler and DTI echocardiography with the use of Vivid 5GE echocardiography device with a 2.5 MHz transducer.
- The Wilkins score was used to assess the suitability of the mitral valve's morphology for balloon mitral valvuloplasty. This scoring system assigns a point value from 1 to 4 for each of: (1) Valve calcification, (2) leaflet mobility, (3) leaflet thickening, and (4) disease of the subvalvular apparatus.[8]

2.4. Severity of mitral stenosis was assessed by

- Mitral valve area was calculated by both the pressure half-time method and planimetry.[6]
- Peak and mean diastolic transmitral gradients were measured by continuous-wave Doppler echocardiography.
- Continuous wave Doppler is used to estimate pulmonary artery systolic and mean diastolic pressure by measuring the peak retrograde pressure drop across the tricuspid valve (Tricuspid regurgitation) and pulmonary valve (Pulmonary regurgitation), respectively. Tricuspid valve velocities can be obtained from parasternal short or long axis projection and apical view whereas pulmonary valve velocities are obtained from the parasternal short axis views (Fig. 1). The pressure drop (gradient) is calculated from the flow velocity using the modified Bernoulli formula ($\Delta P = 4V^2$).[9]
- The maximum peak TR velocity (V) was used to determine right ventricular systolic pressure (RVSP) with the simplified Bernoulli equation which is the sum of the transtricuspid gradient and right atrial pressure (RAP) {RVSP = $4V2$ + RAP}. V is the maximal velocity of the tricuspid regurgitant jet). We further assumed a right atrial mean pressure of 10 mmHg in patients, based on the absence of inferior vena cava dilation greater than 20 mm.[11]

In the absence of a gradient across the pulmonic valve or RVOT, SPAP is equal to RVSP.[12]

2.5. Assessment of RV function

2.5.1. Using M-mode

The tricuspid annular plane systolic excursion (TAPSE) was measured by the level of systolic excursion of the lateral tricuspid valve annulus towards the apex in the four chamber view[4] (see Fig. 2)

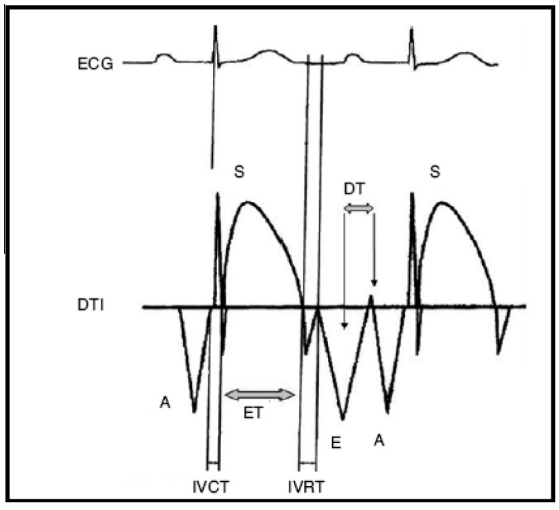

Figure 1 Tissue Doppler echocardiography (DTI) time intervals measured from the tricuspid lateral annulus. ECG: Electrocardiogram; IVCT: isovolumic contraction time; IVRT: isovolumic relaxation time; S: systolic velocity; E\: early diastolic velocity; A\: late diastolic velocity; ET: ejection time; DT: E-wave deceleration time.[10]

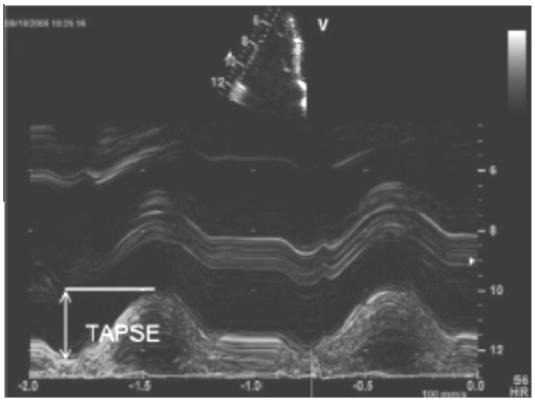

Figure 2 TAPSE.[13]

2.5.2. Doppler tissue imaging

In the apical four-chamber view, the DTI cursor was placed at the tricuspid annulus of the RV free wall.

Three major velocities were recorded: the positive systolic velocity *S* when the tricuspid ring moved towards the cardiac apex, and two negative diastolic velocities $E^{\backslash}$ and $A^{\backslash}$ when the mitral and tricuspid annulus moved towards the base away from the apex. Systolic indexes of DTI included myocardial peak velocity of *S*, IVCT (from the onset of ECG QRS to the beginning of *S*), IVRT: isovolumic relaxation time; the time interval occurring between the end of *S* and the onset of $E^{\backslash}$ wave and ET (from the beginning to the end of S-wave). Diastolic indexes included $E^{\backslash}$ and $A^{\backslash}$ peak velocities.[10]

The Tei index of RV myocardial performance was calculated as the time between tricuspid valve closure and tricuspid valve opening, divided by the RV ejection time and obtained from the tricuspid annulus as the sum of IVCT and IVRT divided by ET[3]

$$\text{Tie index} = \frac{\text{IVCT} + \text{IVRT}}{\text{ET}}$$

Peak myocardial velocities during systole and diastole together with the isovolumic contraction velocity will be measured.[11]

The previous echocardiographic parameters were repeated 24–48 h post BMV and after 3 months for follow up.

2.6. Exclusion of other valvular lesion

2.6.1. Exclusion of complications after PBMV

Percutaneous balloon mitral valvuloplasty was performed using the antegrade double balloon technique

- Balloon size was chosen according to the mitral annular diameter (measured by echocardiography) as follows: sum of diameters of the two balloons equals mitral annular diameter.[14]
- All antegrade approaches begin with the crucial first step of successful trans-septal catheterization with successful access to left atrium through proper part of the atrial septum to allow easy access to the mitral valve then placement of a Mullins type dilator and sheath into LA.[15]
- The two balloon-dilating catheters were then advanced over each of the guide-wires and positioned across the mitral valve parallel to the longitudinal axis of the left ventricle and the Mullins sheath dilator system was removed. The balloon valvotomy catheters were then inflated by hand until the indentation produced by the stenotic mitral valve is no longer seen. Generally one but occasionally two or three inflations were performed. After complete deflation the balloons were removed sequentially.[17]
- Right and left heart haemodynamic data were recorded before and after the procedure. The results were assessed in the cath. lab by invasive measurement of the mean diastolic pressure gradient across the mitral valve.[14]
- Echocardiography was done at the end of the procedure to ensure successful balloon dilatation and to exclude complications as perforation, more than II/IV MR and an atrial left-to-right shunt using colour flow Doppler.
- Successful balloon mitral valvuloplasty criteria are:
  - 50–60% reduction in transmitral gradient.
  - MVA greater than 1.5 cm^2.
  - A decrease in left atrial pressure to less than 18 mmHg in the absence of complications.[7]
  - All of the above criteria were used to assess the success of the procedure.

2.7. Statistical analysis

Statistical presentation and analysis of the present study were conducted, using the mean, standard error, paired *t*-test, Chi-square and Linear Correlation Coefficient and by SPSS V17.

3. Results

The study included 30 patients {5 males (16.67%) and 25 females (83.33%)}.

3.1. Age

Patients' age ranged from 16 to 54 (mean 30.10 ± 9.286 years).

3.2. Weight

Patients' weight ranged from 52 to 68 kg (59.53 ± 3.821).

3.3. Height

Patients' height ranged from 150.00 to 168.000 cm (158.400 ± 3.821).

All the patients were in sinus rhythm with no history or signs of hypertension, diabetes mellitus or lung disease. Majority of patients were in NYHA class II (22 patients; 73.33%) and (8 patients; 26.66%) in NYHA class III.

3.3.1. Transthoracic echocardiography data

Ejection fraction was normal in all the patients, There was no tricuspid regurgitation more than grade II (that remained at the same grade post BMV) or pulmonary valve disease (regurgitation or stenosis) or mitral regurgitation more than grade II. The mean Wilkin's score was 7.76 ± 0.31. The mean MVA by planimetry was 0.893 ± 0.297 cm^2 and by PHT was 0.918 ± 0.221 cm^2. There was a significant correlation between MVA by planimetry and MVA by PHT. The mean

transmitral pressure gradient was 13.967 ± 5.38 while the peak was 27.00 ± 7.551 mmHg.

The mean value of pulmonary systolic arterial pressure (SPAP) was 63.833 ± 19.855 mmHg. Mean TAPSE was 22.32 ± 4.0 mm.

3.3.2. By using tissue Doppler

Mean isovolumic contraction velocity (IVV) was 11.19 ± 4.21 cm/s, mean *S* velocity was 12.78 ± 3.21 cm/s, $E^{\backslash}$ velocity was 9.397 ± 2.385, $A^{\backslash}$ velocity was (12.962 ± 3.009) cm/s and Tie index was (0.502 ± 0.093).

No atrial or atrial appendage thrombus was detected in all patients before the procedure by using TEE. Reassessment of mitral valve score and evaluation of MR was also done by TEE. Balloon size was chosen according to mitral valve annulus measurement by TEE.

Successful BMV was done for all selected patients without any evidence of cardiac perforation and no more than grade II mitral regurgitation was detected by echo Doppler.

There was significant increase in MVA between before and after 24 h where $t = 17.610$ and P-value < 0.001 and by PHT $t = 20.152$ and P-value < 0.001 There was significant increase between before and after 3 ms where $t = 20.920$ and P-value < 0.001 and by PHT $t = 24.414$ and P-value < 0.001. There was no significant change between the area 24 h after and the area either by planimetry or PHT after 3 ms, where $t = 0.931$ and P-value $= 0.360$ and $t = 0.618$ and P-value $= 0.541$ respectively.

There was significant drop in mean and peak PG before and 24 h post BMV where $t = 3.325$ and P-value $= 0.002$; $t = 11.095$ and P-value < 0.001 respectively. There was significant drop in mean PG and peak PG before and after 3 ms where $t = 3.379$ and P-value $= 0.002$; $t = 11.179$ and P-value < 0.001 respectively. There was significant drop in mean PG and peak PG 24 h post BMV and after 3 ms where $t = 1.033$ and P-value $= 0.310$ and by $t = 0.834$ and P-value $= 0.411$ respectively.

3.4. Systolic pulmonary artery pressure (SPAP)

Table 1 showed significant drop in SPAP before and after 24 h where $t = 8.162$ and P-value < 0.001. There was significant drop in SPAP before and after 3 ms where $t = 11.965$ and P-value < 0.001. There was no significant change in SPAP between 24 h after the procedure and 3 months later where $t = 1.489$ and P-value $= 0.147$.

Table 2 shows a significant drop in TAPSE before and 24 h after;before and 3 ms after) the procedure where $t = 2.831$ and P-value $= 0.008$; $t = 3.593$ and P-value $= 0.001$ respectively. There was no significant change 24 h and 3 ms after the procedure where $t = 17.610$ and P-value $= 0.220$.

There was no significant change in IVC flow before and after 24 h where $t = 1.251$ and P-value $= 0.221$.There was no significant drop between before and after 3 ms where $t = 2.028$ and P-value $= 0.062$ .There was no significant change between 24 h and 3 ms after the procedure where $t = 1.139$ and P-value $= 0.264$.

No significant drop was found in *S* velocity (before and 24 h after;before and 3 ms after) where $t = 0.785$ and P-value $= 0.439$; $t = 1.380$ and P-value $= 0.178$ respectively but a slight significant drop occurred 24 h and 3 ms after the procedure where $t = 2.068$ and P-value $= 0.048$.

No significant increase was found in $E^{\backslash}$ velocity (before and 24 h after; before and 3 ms after) where $t = 2.820$ and P-value $= 0.009$; $t = 2.440$ and P-value $= 0.021$ respectively but there was no significant increase 24 h and 3 ms after the procedure where $t = 1.797$ and P-value $= 0.083$.

There was no significant drop in $A^{\backslash}$ velocity (before and 24 h after; before and 3 ms after) where $t = 0.675$ and P-value $= 0.505$; $t = 0.349$ and P-value $= 0.729$ respectively but a slight significant drop occurred 24 h and 3 ms after the procedure where $t = 2.961$ and P-value $= 0.006$.

Table 3 shows a significant drop in Tie index (before and 24 h afterbefore and 3 ms after) where $t = 2.850$ and P-value $= 0.008$; $t = 2.801$ and P-value $= 0.009$ respectively but no significant drop occurred 24 h and 3 ms after the procedure where $t = 0.904$ and P-value $= 0.373$.

Table 4 showed significant correlation between MVA by planimetry with (MVA by PHT, mean pressure, SPAP, E&A) and no relation with the other parameters.

Table 5 showed significant correlation between Tie index with (SPAP, TAPSE and $E^{\backslash}$ vel.) and no relation with the other parameters.

4. Discussion

In the current study, the above mentioned parameters were compared before BMV, (24–48 h) post BMV and 3 m after the procedure to assess the impact of successful BMV on right ventricular functions.

In the current study, SPAP showed a significant decrease between (before and after 24 h; before and after 3 ms); but no significance between (after 24 h and after 3 ms) occurred due to a sudden increase in the mitral valve area which caused a sudden decrease in the pressure gradient across the mitral valve leading to a decrease in left atrial pressure, pulmonary venous congestion and pulmonary artery pressure. This causes acute decrease in RV afterload which has a drawback on the contractile function of RV. Also in the current study SPAP showed significant negative correlation with MVA ($r = -0.542$, $P = 0.002$) after BMV. As well, there was a positive correlation between Tie index and systolic pulmonary artery pressure. This was concordant with the study by Drighil et al.[11] which included 21 patients who demonstrated a significant decrease in SPAP post BMV and a positive correlation between Tie index and pulmonary artery systolic pressure before BMV. Also, Arat et al.'s[10] study on 56 patients who underwent PMBV for isolated rheumatic MS with assessment of RV function before PMBV, 48 h and 3 months after PMBV noted that SPAP was still relatively higher in the intermediate follow up period in patients with baseline PAH compared to patients without baseline PAH, but the difference was not statistically significant.

The decline in SPAP after the procedure was attributable to decreased afterload inpatients with baseline PAH, while patients without PAH exhibited increases in systolic function parameters even though their afterload values seemed to be much lower. It has been reported that pulmonary pressure usually normalizes within 6 months, but may stay elevated for more than 2 years in some patients 10.

Table 1 Change in SPAP (mmHg) before and after BMV.

	SPAP			Paired *t*-test	
	Range	Mean ± SD		*t*	*P*-value
Before	35.00–120.00	63.833 ± 19.855	Before–After 24 h	8.162	< 0.001*
After 24 h	20.00–85.00	39.000 ± 14.916	Before–After 3 ms	11.965	< 0.001*
After 3 ms	20.00–85.00	36.333 ± 12.383	After 24 h–After 3 ms	1.489	0.147

Table 2 Change in TAPSE (mm) before and after BMV.

	TAPSE			Paired *t*-test	
	Range	Mean ± SD		*t*	*P*-value
Before	10.00–32.00	22.320 ± 4.005	Before–After 24 h	2.831	0.008
After 24 h	15.00–28.00	20.657 ± 2.878	Before–After 3 ms	3.593	0.001
After 3 ms	15.00–24.00	20.167 ± 2.493	After 24 h–After 3 ms	1.255	0.220

Table 3 Change in Tie index before and after BMV.

	Tie			Paired *t*-test	
	Range	Mean ± SD		*t*	*P*-value
Before	0.320–0.70	0.502 ± 0.093	Before–After 24 h	2.850	0.008*
After 24hr	0.300–0.61	0.463 ± 0.086	Before–After 3 ms	2.801	0.009*
After 3 ms	0.300–0.61	0.455 ± 0.082	After 24 h–After 3 ms	0.904	0.373

Table 4 Correlation between MVA by planimetry and other parameters before BMV.

	MVA by planimetry	
	r	*P*-value
MVA by (PHT)	0.909	< 0.001
Mean pr.	−0.658	< 0.001
Peak pr.	−0.036	0.851
SPAP	−0.542	0.002
TAPSE	0.122	0.521
IVV	0.255	0.174
S	0.137	0.469
E\	0.436	0.016
A\	0.436	0.016
Tie	−0.135	0.476
Wilkin's score	−0.167	0.377

Table 5 Correlation between Tie index and other parameters before MV.

	Tie	
	r	*P*-value
MVA by (PHT)	−0.052	0.784
Mean pr.	0.073	0.701
Peak pr.	−0.031	0.871
SPAP	0.444	0.014*
TAPSE	−0.390	0.033*
IVV	−0.280	0.133
S	−0.007	0.971
E\	−0.333	0.072*
A\	0.130	0.494
Wilkin's score	0.105	0.581

TAPSE is one of the parameters which reflect systolic function of RV. In the current study TAPSE showed significant decrease between (before and after 24 h; before and after 3 ms); but no significance occurred between after 24 h and after 3 ms; and shows non significant relation with MVA ($r = 0.122$, $P = 0.521$). This was concordant with the study by Mahfouz et al.[16] which included 147 patients in rheumatic MS. This study revealed a significant decrease in TAPSE post BMV denoting that the RV function is usually impaired (even subclinical) in those patients inspite of mild pulmonary hypertension. In accordance with these results, Drighil et al.[11] suggested that patients with MS have depressed global and regional RV function compared with normal subjects; findings go hand in hand with previous radionuclide and haemodynamic studies.

On the other hand, Adavane et al.'s[18] by a study on 33 patients with MS before BMV, 24–48 h after BMV and 1 month after BMV assessed echocardiographic parameters of RV function which showed a significant increase of TAPSE ($P = 0.01$ immediately after BMV; $P < 0.001$ at 1 month) and a significant correlation with the decrease in PVR, RV-RA pressure gradient, the immediate decrease in MVG, and the increase in MVA at one month. In patients with MVA > 1.5 cm^2 after BMV, successful BMV results in a significant improvement of RV systolic function assessed by TAPSE.

In the current study, isovolumic contraction velocities (IVV) on the lateral side of the tricuspid annulus showed no significant decrease between before and 24 h post BMV. There was no significant decrease between before and after 3 months and (24 h and 3 ms) after BMV. Also, there was no significant correlation with MVA ($r = 0.255$, $P = 0.174$) and a negative correlation with SPAP ($r = -0.372$, $P = 0.043$). In contrast, Tayyareci et al. (2008)[3] study on 112 patients with rheumatic MS, found IVV to be significantly decreased and did not correlate with those traditional indices.

In the current study, *S* velocity showed no significant drop in (before and 24 h after) and (before and 3 ms after) with a slight significant drop (24 h and 3 ms) after the procedure.

On the other hand Tayyareci et al.[3] study found *S* velocity to be significantly decreased with no correlation with the other parameters. This was not concordance with Drighil et al.[11] study where S velocity remained unchanged and revealed the reason why systolic velocities during ejection period at the lateral tricuspid annulus remained stable after PTMC, whereas IVA and isovolumic contraction velocity decrease may be related to the fact that parameters illustrating isovolumic contraction period may be more sensitive markers of myocardial function than systolic velocities during the ejection period.

On the other hand, the study by Arat et al.[10] showed that the peak systolic (*S*) velocity of the lateral tricuspid annulus did not differ between the two groups (pts with PAH and pts with no PAH) at baseline. In patients with pulmonary artery hypertension (PAH), it showed a slight increase at 48 h, but fell behind the baseline at 3 months. In patients without PAH, it showed a significant increase at 48 h and remained unchanged at 3 months.

A change in the RV S-wave velocity only in patients without PAH, which might be due to a more protected RV function in this particular group. Another confounding factor might be the shortness of the follow-up period, which was only 3 months. Similar RV changes might be expected within a longer follow-up period in patients with baseline PAH, but this needs to be clarified by further studies 10.

In the current study, $E^{\backslash}$ velocity showed a significant increase in (before and 24 h after) and (before and 3 ms after) with no significant increase 24 h and 3 ms after the procedure. $A^{\backslash}$ velocity showed no significant drop in (before and 24 h after) and (before and 3 ms after) with a slight significant drop 24 h and 3 ms after the procedure .This was concordant with Mahfouz et al.[16] where their study showed the same result. On the other hand Arat et al.[10] study showed that E- and A-wave peak velocities did not differ significantly at 48 h and 3 months.

On the other hand Mehta et al.[19] noticed a non-significant increase in RV free wall annular velocities in 25 patients studied following PBMV and concluded that RV dysfunction persists in the period immediately following BMV despite significant changes in pulmonary artery pressures, and this could explain the persistence of right sided congestion in some of these patients.

The previous two studies were relatively disconcordant with the current study.

Diastolic functions of RV may deteriorate in the presence of normal systolic functions in symptomatic patients with isolated mitral stenosis.[20] Good correlation between RV diastolic dysfunction (and not systolic dysfunction) and increased pulmonary artery systolic pressure has been demonstrated indicating RV systolic dysfunction to be the result of pulmonary hypertension, disturbance of myocardial microcirculation and myocardial fibrosis. These findings suggest that RV diastolic dysfunction is the first detectable myocardial manifestation of inflammatory process.[21]

In the current study, Tie index showed a significant drop (before and 24 h post BMV; before and 3 ms after BMV) but no significant drop occurred (24 h and 3 ms) after BMV. Also, Tie index showed no significant correlation with changes in MVA. As well, there was a positive correlation between Tie index and systolic pulmonary artery pressure. This result was concordant with Drighil et al.[11]. On the other hand, Arat et al.[10] showed that, the baseline Tie index was higher in patients with PAH ($P = 0.004$) and no significant difference in the Tie index during 3-month follow-up. Evaluation at 3 months showed that the Tie index of patients with baseline PAH decreased to values similar to those of patients without baseline PAH. In contrast, Mohan et al.[21]studied 25 consecutive patients with isolated rheumatic mitral stenosis before, immediately after (mean, 40 ± 12 h) and at a mean follow-up of 11.5 months after PTMC. The Tie index was not affected immediately after successful PTMC, however, at follow-up of about one year, the Tie index showed a significant decrease. The Doppler index of combined right ventricular function was significantly correlated with the mean pulmonary artery pressure and systolic pulmonary artery pressure before PTMC and also immediately after the procedure; however, at follow-up, the index had no correlation with the Doppler estimated pulmonary artery systolic pressure.

The drop in RV Tie index and systolic pulmonary artery pressure together immediately post-BMV suggest that RV outflow tract systolic function improved as a result of an acute decrease in RV afterload. This is concordant with the study by Borges et al.[22] who demonstrated an improvement in Tie index after vasodilator therapy in patients with chronic pulmonary hypertension Drighil et al.[11]

5. Conclusion

The current study showed a significant improvement in both systolic and diastolic function of RV as observed by different echocardiographic parameters post BMV in patients without organic TV disease.

6. Recommendations

There is a need to measure RV ejection fraction (RVEF) and RV fractional area. Also RV outflow tract fraction shortening (RVOTfs) should be measured as it reflects the infundibular function.

Isovolumic acceleration (IVA) should be measured as it correlates positively with global LV and RV contractility.

Further work using larger numbers of patients is needed to confirm our findings and to assess their utility in patient's follow-up and management.

7. Limitation

The main limitation of the current study is the small number of patients examined. This was due to the exclusion criteria,

which aimed to limit the number of confounding factors that might interfere with RV function. Another limitation is the short term follow up.

Further studies with large patient groups and long-term follow-up are needed for assessment of the clinical effects of these changes on RV functions.

Conflict of interest

None.

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